YELLOW FEVER IN THE GOLD COAST: ITS ENDEMIC AND EPIDEMIC CHARACTER

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The recently reported cases of yellow fever in different parts of the Gold Coast* bring into prominence again the subject of its endemicity in West Africa, and suggest a consideration of the conditions which give rise to these periodic outbreaks.

That the West Coast of Africa has been an endemic centre no one disputes, but opinion is divided upon the question of the presence of yellow fever as an endemic disease to-day.

The Yellow Fever Commission (West Africa) appointed by the Colonial Office in 1913 considered the evidence submitted was in favour of the belief that West Africa is an endemic centre (1916). More recently, Guiteras (1921) and Hoffman (1921) expressed the opinion that this centre has already ceased to exist. This supports the view held by Ross, who suggests that the Stegomyia mosquito is not sufficiently numerous to permit the disease to maintain itself.

The investigations of the Rockefeller Foundation in 1920 failed to discover a single case, and we find that out of nearly half a million recorded illnesses treated in Nigeria during the years 1919-21, there is but a single diagnosis of yellow fever.

In spite of this formidable array of opinion and facts, it is not possible to ignore the significance of the existence of a disease which does not conform to prevalent types of fever and which is capable of being diagnosed as yellow fever.

The reports of the recent cases occurring among Europeans on the Gold Coast leave very little room for doubt as to the accuracy of the diagnosis. In all these were the black tarry or coffee grounds vomit, a marked diminution in the quantity of urine—amounting in

* A cable received at the Colonial Office on July 11, 1923, stated that 25 cases of yellow fever had occurred in the Gold Coast since November 1, 1922—18 in Europeans, all fatal, and 7 in natives, 2 fatal.

some cases to complete anuria—with albuminuria, associated with symptoms of extreme urgency followed rapidly by death. When post-mortem examinations were made, fatty degeneration changes were found in the liver and kidney. These features were common to all, but haemorrhage from mucous membranes, conjunctival injection, varying degrees of jaundice, Faget's sign and high fever are described. It is also noteworthy that, with one exception, the fatal termination occurred within a week of the onset of the illness.

In the face of such evidence, it must be admitted that yellow fever is a disease of West Africa. The next important point is—in what form does it exist?

An examination of the trade routes between the West Coast of Africa and other parts of the world fail to supply us with an external source of infection. In the 1911 outbreak at Bathurst it was thought that the occurrence of the disease at this port was connected with the visit of the s.s. 'Akassa.' Subsequent enquiries, however, failed to confirm this. In the epidemic under consideration, there is not the slightest ground for suspecting that the disease was introduced from without. It seems, then, that we must assume that yellow fever does exist in West Africa, in some latent form requiring special conditions for its development.

That special conditions are required may be gathered from the infrequency with which the disease occurs among Europeans in any one place, notwithstanding that the essentials for the maintenance and spread of the disease are always present—the virus, the carrier and the non-immune.

The explanation which used to be given for the comparative freedom from yellow fever among Europeans on the West Coast of Africa was, that the newcomer usually became the subject of a mild infection which conferred immunity for the remainder of his stay in the country. But this contention cannot be maintained, in view of the fact that victims of the disease have succumbed to it after periods of residence up to thirty years. It is necessary, therefore, to look for some other explanation.

When an endeavour is being made to discover the aetiological factor concerned in the outbreak of an infectious disease, it is helpful to be able to note the conditions under which it has died out in certain places.

Peterson's (1922) view of the spontaneous elimination of yellow fever in St. Thomas—which is based upon Carter's principle of 'the failure of the human host'—suggests that the virus gradually becomes attenuated until it reaches the point of extinction. In other words, the commerce between the mosquito and the unresponsive native eradicates the disease, not by the process of conferring general immunity, but by the death of the virus. The logical inference which may be drawn from this is, that the West Coast of Africa would cease to be a yellow fever area as soon as, or shortly after, less immune individuals were excluded from the country. The European is, of course, the obvious non-immune. Yet if we study the records of yellow fever epidemics, we shall find long intervals between outbreaks in any one place, and that they are traceable to a non-European source. This is fair presumptive evidence that the European is an accidental victim rather than an agent in maintaining the disease.

It may be urged that mild cases occur among Europeans which escape diagnosis. The mortality rate in the 1910-11 outbreaks (1913), which show six recoveries in forty cases, is against this; more especially when the reports of these recovered cases are scrutinised.

Of the six, three are included without any details of their illness, and as such, merely indicate people who were sick at the time.

Of the three remaining, two are so untypical of yellow fever, that it is safe to presume their inclusion was due to the prevalence of the disease rather than to the character of their physical signs and symptoms. The last case of this group appears to have responded, eventually, to energetic treatment with quinine. Albuminuria was absent throughout the illness.

In the present epidemic in the Gold Coast, the mortality among the Europeans attacked is 100 per cent. If we exclude the six doubtful cases referred to above, it would appear that yellow fever in West Africa is invariably fatal to Europeans. At any rate, it may be said it shows itself in a way which is not likely to be overlooked.

To recapitulate: The indigenous native, dissociated from the presence of newcomers, is incapable of maintaining the virus. The disease is not overlooked when it occurs among Europeans. The intervals which elapse between outbreaks among Europeans are proof

that the European is not responsible for the fact that yellow fever is an endemic disease in West Africa.

It is obvious then that some other section of the community keeps the virus alive in a 'larval' state.

It has been said that previous recorded outbreaks are traceable to a native source. In the Reports of the 1910-11 epidemic (1913) it will be found that in every case when mention is made of living conditions, there is the association of close native proximity to the European attacked. Further it will be found that the native element is an imported one, either from the confines of the colony itself, or from a more remote part of West Africa.

Cases numbers 43 and 44 were Kroo-boys. 37 was a Yoruba lately come to Lagos, where he was taken ill. 29 and 38 also occurred in Kroo-boys. 28 was a Hausa. 19 and 20 were of the Mendi tribe living in Freetown at the time they were taken ill.

The foregoing comprise all these cases among natives where it is possible to identify their nationality. The remainder come under the, not very illuminating, description 'A native born and bred in West Africa.'

In the Gold Coast epidemic of this year, the cases at Saltpond were traceable to a Kroo-boy who died of yellow fever shortly after his arrival there. Later, eighteen miles away, Cape Coast was attacked, isolated cases subsequently occurring at Winnebal, Accra, Keta and Seccondee.

On the Gold Coast we find that West Africans who come to the Colony pursue one of the following callings:—Trading, soldiering, mining, as railway and road labourers, or manual work at the seaports. With the exception of the last-named class, it will be found that the principle of segregation is conformed with. The migratory trader, the Hausa, has his Zonga to go to, the soldier his barracks, while railway and road labourers (usually drawn from the Northern Territories) have carefully supervised camps.

The porter of the Gold Coast, the Kroo-boy, has no such provision made for him, and he is to be found, as a rule, domiciled in the compound of his employer, usually a European. He also has access to the native quarters of the town, and consorts with other Kroo boys who are employed by native merchants. There is here, then, a connecting link between the two classes which have been

excluded as not being responsible for maintaining the disease. The Kroo-boys also represent, numerically, a section of the community which must be taken into consideration, and they become suspects, partly because the process of elimination adopted here has left them unexonerated, and also for the reason that they are known to contract the disease and to have been responsible for outbreaks among Europeans.

The question now arises, how is it that these Kroo-boys are more susceptible to infection than the indigenous native?

It will be shown later that the immunity to yellow fever with which all West Africans are endowed is merely relative in degree and breaks down under certain conditions. It is suggested here that one such condition is change of environment.

Apparently the native's degree of immunity is sufficient so long as he remains in his own country, but becomes impaired when he goes to live in another part of West Africa. There is support for this assumption in the fact that West Africans do contract 'fever' when they leave one part of West Africa to take up residence in another. This has been recognised by Government Medical Officers, and the West Africans themselves are aware of it. An opportunity of observing this phenomenon occurs when a native official is transferred to a new station, or is on leave from a Colony of which he is not a native. That the 'fever' mentioned above is often due to malaria, there is very little doubt. An intensive and fatal case of this disease was seen at Accra in a Kroo-boy who had been resident in the Colony for six months. It is known that malaria does not attain to such severity in the adult indigenous population of West Africa. The influence of environment is, therefore, a factor which must be reckoned with when the subject of immunity is being considered.

From these premises it is easy to reconstruct the sequence of events. The Kroo-boy who migrates to some other part of West Africa automatically becomes more susceptible to yellow fever. He contracts the disease in a mild form, unrecognisable as such, and causing very little, if any, inconvenience. But an impetus has been given to the virus which, under favourable conditions, ultimately becomes so enhanced as to give rise to definite illness. The final stage is reached when living conditions make it possible for the

Kroo-boy to pass on this infection to a European. When this occurs the virus has attained to a very virulent degree of toxicity, which if unchecked by the wholesale destruction of the mosquito, will be capable, ultimately, of infecting—sometimes with fatal consequences—the indigenous and erstwhile unsusceptible native. This happened both at Saltpond and Cape Coast during the present epidemic, and is proof of what has already been said, that the West African's immunity is merely a relative one.

It is well known that any break in the chain of essentials which go to produce a yellow fever infection is sufficient to stop, or at least interrupt, the process. The lapse of time which takes place between observed epidemics in West Africa, seems to suggest that the chain is delicate in its construction and that the process of building up the virus sufficiently to produce recognisable effects is a long one. Segregation of Europeans—in as far as it obtains in West Africa—appears to have the effect of lengthening the process. It has certainly provided immunity for the segregated, for in no single instance has a case occurred among them. When it is remembered that in segregation areas native servants—often Kroo-boys and natives of the Northern Territories—live in close contact with the European, it would seem that the slightest precautions are sufficient to prevent infection. As Carter suggests in his statement of requirements for the maintenance of a yellow fever infection, the number of mosquitoes may fall short of what is necessary. It probably will be found also, that non-interference with the mosquitoes, overcrowding and lack of light and ventilation are necessary. Routine sanitary work probably intereferes from time to time with one or other of these subsidiary requirements, and has the effect of delaying the development of the virus. But sooner or later, it would seem, an area escapes over a period which permits it to become intensely infective, and an outbreak of yellow fever results.

The localised character of these outbreaks in a town is due to the well-marked domestic habits of the mosquito concerned. If we look at the spot maps accompanying the Reports of the 1910-11 epidemic, it is easy to see the human agency which carries the disease from one part of a town to another, over distances which leave intermediate areas unattacked.

It is, therefore, the infected rather than the infective element

which is responsible for the spread of the disease. If we can control the former and keep it from coming into close living contact with the unsegregated European, there is a reasonable prospect of preventing re-occurrences of these outbreaks. Efforts at controlling the other element have hitherto met with very little appreciable success. That temporary success is obtainable has been amply demonstrated during the present epidemic. Towns where the Stegomyia index is normally 80 per cent. have, after a week's intensive work, had this figure reduced to below 5 per cent. The means employed, other than fumigation of the area in which cases occurred, were the usual mosquito brigades under the supervision of European volunteers.

The effectiveness of this measure, when considered in the light of what has already been said, suggests the advisability of instituting a 'cleaning-up week' at least once a year, in every town where cases of yellow fever have been known to occur within the last twenty years. It should also be a matter of routine that when a case of yellow fever is reported in a Colony, every town with which the infected area is connected by road, rail, or sea, should immediately start energetic anti-stegomyia measures. This will prevent outbreaks elsewhere, for the reason that the number of mosquitoes remaining will not be able to maintain the disease. At any rate, the possibility of a secondary focus being established will be a very remote one. In the intervals between epidemics, Government Medical Officers and other Medical practitioners should be asked to observe carefully cases of fever which occur in West Africans who are strangers in the place, with a view to early diagnosis, thus ensuring prevention of the development and spread of the disease.

REFERENCES

Guiteras, Juan (1921). Observations on Yellow Fever in a Recent Visit to West Africa. *Sanidad y Beneficencia*, Vol. XXV, p. 21.

Hoffmann, W. H. (1921). Das Gelbfieber in Westafrika. *Arch. f. Schiffs-u. Trop.-Hyg.*, Vol. XXV, p. 291.

Peterson, E. (1922). Yellow Fever in St. Thomas, with special reference to its spontaneous elimination. *U.S. Naval Medical Bulletin*, Vol. XVII, p. 555.

Report on Certain Outbreaks of Yellow Fever in 1910 and 1911. Waterlow & Sons, London (1913).

Yellow Fever Commission (West Africa) Fourth and Final Report. J. and A. Churchill, London. (1916), p. 255.